ORIGINAL ARTICLE

Carotid Artery Ultrasonographic Assessment in Patients from the Fremantle Diabetes Study Phase II with Carotid Bruits Detected by Electronic Auscultation

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Abstract

Background: Electronic auscultation appears superior to acoustic auscultation for identifying hemodynamic abnormalities. The aim of this study was to determine whether carotid bruits detected by electronic stethoscope in patients with diabetes are associated with stenoses and increased carotid intima-medial thickness (CIMT).

Subjects and Methods: Fifty Fremantle Diabetes Study patients (mean $\pm$ SD age, 73.7 ± 10.0 years; 38.0% males) with a bruit found by electronic auscultation and 50 age- and sex-matched patients with normal carotid sounds were studied. The degree of stenosis and CIMT were assessed from duplex ultrasonography.

Results: Patients with a bruit were more likely to have stenosis of $\geq 50\%$ and CIMT of >1.0 mm than those without (odds ratios [95% confidence intervals] = 14.0 [1.8–106.5] and 5.3 [1.8–15.3], respectively; both $P=0.001$). For the six patients with stenosis of $\geq 70\%$, five had a bruit, and one (with a known total occlusion) did not (odds ratio = 5.0 [0.6–42.8]; $P=0.22$). The sensitivity and specificity of carotid bruit for stenoses of $\geq 50\%$ were 88% and 58%, respectively; respective values for stenoses of $\geq 70\%$ were 83% and 52%. The equivalent negative predictive values were 96% and 98%, and positive predictive values were 30% and 10%, respectively.

Conclusions: Electronic recording of carotid sounds for later interpretation is convenient and reliable. Most patients with stenoses had an overlying bruit. Most bruits were false positives, but ultrasonography is justified to document extent of disease; CIMT measurement will identify increased vascular risk in most of these patients. The absence of a bruit was rarely a false-negative finding, suggesting that these patients can usually be reassured that they do not have hemodynamically important stenosis.

Introduction

A CAROTID BRUIT DETECTED by conventional acoustic auscultation is associated with a two- to fourfold increased risk of transient ischemic attack, stroke, and death in general population studies.¹ Diabetes further increases the risk of both a bruit² and the subsequent risk of stroke.³ Meta-analysis suggests that the sensitivity and specificity of a bruit for a $\geq 70\%$ carotid stenosis detected by ultrasonography, angiography, and/or oculoplethysmography are only 53% and 83%, respectively.⁴ Nevertheless, current guidelines state that it is reasonable to perform duplex ultrasonography in patients with or without diabetes who have a carotid bruit to determine whether a hemodynamically significant stenosis

is present.⁵ Ultrasonography remains a noninvasive way of identifying which patients should benefit from optimal medical management, including antiplatelet therapy and perhaps further imaging with a view to revascularization.^{6,7} In addition, ultrasonographic measurement of the carotid intima-medial thickness (CIMT) can provide indirect evidence of coexistent coronary artery and/or peripheral vascular disease,^{8–10} further reinforcing the need for optimized cardiovascular risk factor management.

Carotid auscultation using an acoustic stethoscope is a simple tool that requires application of the correct technique in a noise-free environment by a skilled operator who instantaneously interprets what is heard, including differentiation of a bruit from a transmitted cardiac murmur. When

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these conditions cannot be met, the opportunity for potentially valuable screening is lost. The electronic stethoscope has, however, emerged as a way in which a trained nonexpert operator can obtain a permanent auscultatory record for later expert interpretation. These digital recordings are of better quality than those obtained using acoustic stethoscopes.^{11,12} This technology has been applied successfully in clinically challenging situations such as in aviation medicine,¹² prisons,¹³ and remote clinics.¹⁴

The sensitivity and specificity of carotid bruits ascertained by electronic auscultation for ultrasonographically identified carotid stenosis and increased CIMT have not been examined previously. We hypothesized that the use of an electronic stethoscope by a trained nonexpert operator with subsequent evaluation by an experienced clinician would improve the diagnosis of carotid atherosclerosis in patients with diabetes.

Subjects and Methods

Patients

We studied participants in the Fremantle Diabetes Study (FDS) Phase II (FDS2), a community-based prospective observational study conducted in an urban region of Australia inhabited by approximately 153,000 people. A detailed description of FDS2 identification/recruitment procedures, sample characteristics, classification of diabetes type, and details of nonrecruited patients has been published previously.¹⁵ The FDS2 protocol was approved by the Human Research Ethics Committee of the Southern Metropolitan Area Health Service, and all subjects gave informed consent before participation.

Of 1,732 FDS2 subjects recruited between 2008 and 2011, 361 had died or withdrawn by early 2013. Of the 1,371 available for participation in the present substudy, 84 (6.1%) were identified as having a carotid bruit by electronic auscultation at prior FDS2 assessments. Each of these patients and one age- and gender-matched FDS2 subject without a bruit were invited to undergo carotid Doppler ultrasonography. We did not exclude patients who reported a history/symptoms of cerebrovascular disease or those with prior carotid imaging but did exclude those who had undergone carotid revascularization subsequent to identification of the bruit.

Clinical assessment

At FDS2 baseline assessment and subsequent biennial reviews, a comprehensive history of diabetes and comorbidities was recorded, a physical examination was performed by a trained nurse, and fasting blood and urine samples were taken for analyses in a single nationally accredited laboratory.¹⁵ Each patient underwent standardized screening for carotid bruits by electronic auscultation. A Littmann 3000 stethoscope (3M, North Ryde, NSW, Australia) was applied to six areas (upper and lower carotid bilaterally and aortic and pulmonary areas), and a recording of ≥ 5 beats was taken at each site with the patient holding his or her breath in deep inspiration. The recordings were subsequently analyzed by one of two study physicians (B.A.S. or T.M.E.D.) using the Littmann sound analysis software (version 2.0.C). A carotid bruit was considered present if there was a clearly audible systolic bruit in the upper and/or lower carotid region and no infraclavicular murmur. Where interpretation was unclear,

the recording was assessed by the alternate study physician, and a consensus was reached. Other chronic complications were ascertained using standard criteria.¹⁶

Ultrasonography

Carotid duplex ultrasonography was performed using the iU22 system (Philips Healthcare, North Ryde) under Australasian Society for Ultrasound in Medicine guidelines.¹⁷ Images were assessed by two experienced sonographers and two radiologists and categorized as follows: (1) 0% stenosis with normal waveform/image; (2) <15% diameter reduction assessed from deceleration with a spectral broadening and peak systolic velocity (PSV) of <125 cm/s; (3) 16–49% diameter reduction with a pansystolic spectral broadening and PSV of <125 cm/s; (4) 50–69% diameter reduction with pansystolic spectral broadening, PSV of >125 cm/s, and end-diastolic velocity of <110 cm/s or ratio of internal carotid artery PSV to common carotid artery PSV of >2; (5) 70–79% diameter reduction with pansystolic spectral broadening and PSV of >270 cm/s, end-diastolic velocity of >110 cm/s, or ratio of internal carotid artery PSV to common carotid artery PSV of >4; (6) 80–99% diameter reduction and criteria as in (5) plus end-diastolic velocity of >140 cm/s; and (7) occluded with no flow terminal thump.

Bilateral measurements of the anterior, lateral, and posterior bifurcation CIMT were done as per the American Society of Echocardiography consensus statement¹⁸ using a 12.5-MHz linear array transducer. Preprocessing configurations (log gain compensation of 60 dB, image persistence) were kept constant. Using antero-oblique insonation, the far-wall CIMT was adjusted bilaterally at the carotid bifurcation and 1.0 cm proximally from the flow divider by visualizing the double lines of the carotid artery on the near and the far wall of the common carotid artery. Optimized transducer depth (usually 4.0 cm) was adjusted to avoid slice thickness artifacts. The images were captured during systole at the R-waves over three or four cardiac cycles. Three edge-to-edge measurements were taken of the far wall with a CIMT over ≥ 1 cm lengths without the zoom function. The presence of carotid plaque was defined as focal wall thickening of $\geq 50\%$ greater than that of the surrounding vessel wall or as a focal region with the intima-medial thickness >1.5 mm protruding into the lumen and distinct from the adjacent boundary.¹⁸ Plaques ranged from hard (calcified) to soft (echogenic without calcification).¹⁷

CIMT threshold and percentile distribution

A fixed CIMT threshold of ≥ 1.0 mm is considered an adverse cardiovascular indicator.^{19,20} In the ASE consensus statement,¹⁸ the nomogram of the CIMT percentile distribution is taken from the Carotid Atherosclerosis Progression Study,²¹ which classifies CIMT values in percentile distribution by age, sex, and race/ethnicity. Those in the $\geq 75^{\text{th}}$, 25–75th, and $\leq 25^{\text{th}}$ percentile ranges are considered at increased, average, and lower risk, respectively.

Screening for Heart Attack Prevention and Education guidelines

Under Screening for Heart Attack Prevention and Education (SHAPE) guidelines,²² patients are categorized as lower,

moderate, moderately high, high, and very high risk based on age and carotid ultrasound findings. A positive atherosclerotic test is defined as CIMA (worse side) of $\geq 50^{\text{th}}$ percentile or presence of plaque (focal wall thickening $\geq 50\%$ greater than that of the surrounding vessel wall or a focal region with CIMA of >1.5 mm protruding into the lumen and distinct from the adjacent boundary¹⁷). All ≥ 75 year olds are assumed high risk unless they have a carotid stenosis of $\geq 50\%$, which, with a positive test, indicates very high risk. High-risk patients have a positive test, $<50\%$ carotid stenosis, and a CIMA of ≥ 1 mm, a CIMA of $\geq 75^{\text{th}}$ percentile, or carotid plaque. Moderately high-risk patients have a positive test, a CIMA of <1.0 mm, and $<75^{\text{th}}$ percentile with no plaque. Moderate-risk patients have a negative test and cardiovascular risk factors. Those at lower risk have a negative test and no risk factors.

Statistical analysis

Data are presented as proportions, mean $\pm$ SD, geometric mean (SD range), or, for those not conforming to a normal or log-normal distribution, median [interquartile range]. For independent samples, two-way comparisons of proportions were by Fisher's exact test, whereas Student's *t* test was used for normally distributed variables. For comparisons between individually matched cases and controls, McNemar's and McNemar-Bowker's tests as well as matched odds ratio (OR) with 95% confidence interval were used for proportions, paired *t* tests for normally distributed variables, and the Wilcoxon signed-rank test for non-normally distributed variables.²³ A two-sided $P < 0.05$ was considered significant.

Results

Of the 84 patients with a carotid bruit, 50 underwent carotid Doppler ultrasonography, one was ineligible (recent carotid endarterectomy), three declined, and 30 did not respond to the invitation to participate. Compared with these latter 33 patients, the 50 recruits were not significantly different in age or sex distribution ($P \geq 0.54$) but had a significantly shorter median duration of diabetes (11.9 [7.7–20.7] vs. 19.1 [11.2–29.6] years; $P = 0.021$). At the time of ultrasonography, the 50 cases and 50 controls had similar proportions of type 1 and 2 diabetes and similar diabetes duration (Table 1). The cases had a lower diastolic but similar systolic blood pressure to the controls, consistent with a wider pulse pressure, and their serum high-density lipoprotein cholesterol concentrations were higher.

The carotid ultrasound parameters and SHAPE categories are summarized in Table 2. Compared with those without a bruit, patients with a bruit were significantly more likely to have carotid stenosis of $\geq 50\%$ and a CIMA of >1.0 mm (OR [95% confidence interval] = 14.0 [1.8–106.5] and 5.3 [1.8–15.3], respectively; $P = 0.001$ in each case), and they were also more likely to fall within the very high SHAPE vascular risk category (trend $P = 0.018$). For the small numbers detected as having a high-grade carotid stenosis ($\geq 70\%$), those with a bruit were more likely to fall into this category, but this did not reach statistical significance (OR = 5.0 [0.6–42.8]; $P = 0.22$). Three patients in this category—two in the bruit group and the one case in the nonbruit group who also had a history of coronary artery disease and peripheral vascular disease—were aware that they had a completely occluded internal carotid artery on one side prior to the ultrasound.

TABLE 1. CHARACTERISTICS OF THE PATIENTS CLASSIFIED BY CAROTID BRUIT STATUS

	Carotid bruit detected	No carotid bruit detected	P
Number	50	50	
Age (years)	73.7 $\pm$ 10.0	73.8 $\pm$ 10.1	0.95
Sex (% male)	38.0	38.0	1.00
Type 2 diabetes (%)	90.0	96.0	0.38
Diabetes duration (years)	12.9 [8.0–21.0]	13.5 [8.3–20.1]	0.86
HbA _{1c}			
%	6.7 [6.3–7.3]	7.1 [6.4–7.6]	0.17
mmol/mol	50 [45–56]	54 [46–60]	0.17
Body mass index (kg/m ²)	29.1 $\pm$ 4.7	30.6 $\pm$ 5.4	0.14
Supine blood pressure (mm Hg)			
Systolic	149 $\pm$ 21	143 $\pm$ 21	0.20
Diastolic	73 $\pm$ 12	78 $\pm$ 11	0.010
Antihypertensive therapy (%)	90.0	88.0	1.00
Total serum cholesterol (mmol/L)	4.3 $\pm$ 1.0	4.3 $\pm$ 0.9	0.87
Serum HDL-cholesterol (mmol/L)	1.31 $\pm$ 0.33	1.17 $\pm$ 0.28	0.037
Serum LDL-cholesterol (mmol/L)	2.4 $\pm$ 0.9	2.3 $\pm$ 0.7	0.62
Serum triglycerides (mmol/L)	1.4 (0.9–2.1)	1.6 (0.9–2.8)	0.08
Lipid-modifying therapy (%)	76.0	76.0	1.00
Aspirin therapy (%)	44.0	44.0	1.00
Urinary albumin:creatinine (mg/mmol)	2.4 (0.7–8.3)	3.4 (1.0–11.4)	0.14
Estimated glomerular filtration rate <60 mL/min/1.73 m ² (%)	28.0	38.0	0.33
Self-reported			
Ischemic heart disease (%)	28.0	30.0	1.00
Stroke/transient ischemic attack (%)	6.0	12.0	0.51
Carotid artery disease/prior ultrasound (%)	14.0	4.0	0.13

Interquartile range is given in square brackets, and SD range in parentheses.

HbA_{1c}, glycosylated hemoglobin; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

TABLE 2. ULTRASONOGRAPHIC FINDINGS AND SCREENING FOR HEART ATTACK PREVENTION AND EDUCATION CATEGORIES IN THE TWO PATIENT GROUPS

	<i>Carotid bruit detected</i>	<i>No carotid bruit detected</i>	<i>Matched OR (95% CI)</i>	<i>P</i>
Carotid stenosis				
$\geq 50\%$	15 (30.0)	2 (4.0)	14.0 (1.8–106.5)	0.001
$\geq 70\%$	5 (10.0)	1 (2.0)	5.0 (0.6–42.8)	0.22
Hard or mixed plaque	39 (78.0)	26 (52.0)	4.3 (1.4–12.6)	0.007
CIMT				
>1.0 mm	26 (52.0)	9 (18.0)	5.3 (1.8–15.3)	0.001
$\geq 75^{\text{th}}$ percentile ^a	30 (66.7)	20 (44.4)	3.0 (1.1–8.3)	0.041
SHAPE category				
Lower/moderate	1 (2.0)	2 (4.0)		0.018
Moderately high	1 (2.0)	3 (6.0)		
High	33 (66.0)	43 (86.0)		
Very high	15 (30.0)	2 (4.0)		

^a $n=45$ for case/control pairs, both of whom were within the <85 year age range used by the Screening for Heart Attack Prevention and Education (SHAPE) guidelines.

CI, confidence interval; CIMT, carotid intima-medial thickness; OR, odds ratio.

Arterial tortuosity was graded semiquantitatively as minimal, moderate, or marked, and those with a carotid bruit were more likely to have at least minimal tortuosity (OR=2.0 [0.9–4.7]; $P=0.15$) and at least moderate tortuosity (3.3 [0.9–12.1]; $P=0.09$). None of the cases or controls with at least minimal tortuosity had a carotid stenosis of $\geq 70\%$.

The sensitivity, specificity, positive predictive value, and negative predictive value of a carotid bruit for each parameter are detailed in Table 3. The sensitivity of an electronically detected carotid bruit for a stenosis of $\geq 70\%$ was 83% with a modest specificity of 52%. For a stenosis of $\geq 50\%$, the specificity and sensitivity were both higher, but the negative predictive value was $\geq 96\%$ in both cases. For CIMT of >1.0 mm, CIMT of $\geq 75^{\text{th}}$ percentile, and the presence of plaque, sensitivity and specificity ranged between 60% and 74%. After exclusion of eight case/control pairs that included at least one patient who had reported a history of carotid artery disease or prior ultrasonographic assessment prompted by symptoms, the sensitivity of a bruit for a stenosis of $\geq 50\%$

or $\geq 70\%$ increased to 100% but with wider 95% confidence intervals (Table 3).

Discussion

The present data show that carotid bruits identified by an electronic stethoscope in community-based patients with diabetes have high sensitivity ($\geq 83\%$) and negative predictive value ($\geq 96\%$) for both moderate- ($\geq 50\%$) and high-grade ($\geq 70\%$) carotid stenoses detected subsequently by carotid duplex ultrasonography. Given that the positive predictive value was low ($\leq 30\%$), these observations suggest that most of the patients with carotid stenoses had an overlying bruit, but that most bruits were false positives, and that the absence of a bruit was rarely a false-negative finding. The practical implications of these observations are that (1) electronic recording of carotid sounds by a trained operator for later transmission and interpretation appears reliable when expert acoustic auscultation is unavailable, (2) patients with diabetes who have no carotid bruit detected in this way

TABLE 3. SENSITIVITY, SPECIFICITY, POSITIVE PREDICTIVE VALUE, AND NEGATIVE PREDICTIVE VALUE OF CAROTID BRUITS FOR STENOSIS, CAROTID INTIMA-MEDIAL THICKNESS, AND PLAQUE

	<i>Sensitivity</i>	<i>Specificity</i>	<i>Positive predictive value</i>	<i>Negative predictive value</i>
Patients with carotid bruit				
Carotid stenosis				
$\geq 50\%$	88 (62–98)	58 (47–68)	30 (18–45)	96 (85–99)
$\geq 70\%$	83 (37–99)	52 (42–62)	10 (4–23)	98 (88–100)
CIMT				
≥ 1.0 mm	74 (56–87)	63 (50–74)	52 (38–66)	82 (68–91)
$\geq 75^{\text{th}}$ percentile	60 (45–73)	63 (46–77)	67 (51–80)	56 (40–70)
Hard/mixed plaque	60 (47–72)	69 (51–83)	78 (64–88)	48 (34–62)
Asymptomatic cases/controls				
Carotid stenosis $\geq 50\%$	100 (63–100)	56 (44–67)	21 (11–37)	100 (90–100)
Carotid stenosis $\geq 70\%$	100 (20–100)	51 (40–62)	5 (1–17)	95 (90–100)

Data are estimated population midpoints (95% confidence intervals). Data for 42 case/control pairs, both of whom had no prior symptoms of carotid arterial disease, are also shown.

CIMT, carotid intima-medial thickness.

can usually be reassured that they do not have hemodynamically important carotid atherosclerosis, (3) patients with diabetes and a carotid bruit will usually not have a significant ($\geq 70\%$) stenosis, but, consistent with current guidelines,⁵ duplex ultrasonography should still be recommended to document more objectively the extent of disease, and (4) measurement of the CIMT in patients with diabetes and a carotid bruit will identify increased vascular risk in most of these patients regardless of the presence of stenosis.

A recent meta-analysis of studies examining the utility of carotid bruits ascertained using an acoustic stethoscope for clinically significant carotid stenoses ($>70\%$) generated a pooled sensitivity of 53% and a specificity of 83%.⁴ The higher sensitivity and lower specificity found in the present study could be due to several factors. First, the better acoustic quality obtained using the electronic stethoscope^{11,12} may have meant that more bruits were detected. This includes softer bruits both associated with reduced flow in severely stenosed vessels,²⁴ thus increasing sensitivity, and as a manifestation of low-grade stenoses or vascular tortuosity, which would attenuate specificity and positive predictive value. Indeed, the prevalence of bruits detected by acoustic stethoscope in Phase 1 of the FDS (FDS1) between 1993 and 1996 was lower at 4.5%³ compared with 6.1% in the present study. Second, we used relatively rigorous methodology in an attempt to eliminate the confounding influence of transmitted cardiac murmurs²⁴ that would potentially reduce sensitivity. Infraclavicular auscultation was not performed in several studies,^{7,25,26} and, in another, ascertainment of bruits was based solely on the referring physician's assessment.²⁷

The cost-effectiveness of screening for asymptomatic carotid artery stenosis using duplex ultrasonography has not been assessed through a randomized clinical trial. It remains contentious and dependent on key variables such as the stenotic threshold for intervention (which has been set at 50%, 60%, and 70%), the prevalence of stenosis in the target population (which can be increased, for example, by including only older men), and treatment outcomes (especially when assessment and intervention are not carried out in centers of excellence).^{28–30} Indeed the sensitivity and specificity of duplex ultrasonography itself compared with angiography are variable in published studies^{31–33} but typically are $\geq 90\%$ for a stenosis $\geq 70\%$.²⁹ Although a formal health economic evaluation would be required, our data suggest that prescreening with electronic auscultation could improve cost-effectiveness, especially because specificity and negative predictive value were both $\geq 96\%$ in our asymptomatic patients.

There is evidence that carotid bruits are more predictive of cardiovascular than cerebrovascular disease outcomes, including death.^{10,34} CIMT is a proven indicator of cardiovascular risk and can refine assessment of patients at intermediate risk.¹⁸ Our data demonstrate that the presence of a carotid bruit increases the likelihood of a CIMT of ≥ 1 mm threefold. There were also 50% more patients with carotid plaques in the group with detectable carotid bruits. A recently published editorial suggested that a combination of CIMT of ≥ 1 mm and carotid plaque might improve coronary risk prediction more than either parameter alone,³⁵ and the SHAPE guidelines incorporate these variables independently.²² Indeed, almost one-third of our patients with bruits were in the very high SHAPE vascular risk category compared with only 4% of those without bruits. Thus, observations and

measures other than degree of carotid stenosis can inform management because patients assessed at very high risk are recommended to have a stricter serum low-density lipoprotein cholesterol target (<1.8 mmol/L) than other categories and to be considered for investigation of myocardial ischemia such as through an exercise stress test.²² Indeed, most of our patients, including those with a CIMT of ≥ 1 mm and/or a carotid bruit, had a serum low-density lipoprotein cholesterol level of ≥ 1.8 mmol/L despite the fact that the majority were taking lipid-modifying agents (largely statins), suggesting that therapeutic intensification may be possible. The apparently paradoxical higher serum high-density lipoprotein cholesterol concentrations in those with carotid bruits is most likely a chance finding.

There was a trend to increased carotid artery tortuosity in the patients with carotid bruits, especially for at least moderate degrees of this vascular anomaly, which was independent of the presence of high-grade ($\geq 70\%$) stenosis. Mild tortuosity is a relatively common asymptomatic finding,³⁶ but there is debate as to the significance of more severe tortuosity. It has been associated with atherosclerosis and its risk factors, including hypertension,³⁶ and it predicts subsequent intraprocedural complications in patients undergoing carotid artery stenting,³⁷ but its prognostic value for cerebrovascular events is uncertain.³⁸ In the present study, at least moderately severe tortuosity was present in one in five patients with carotid bruit and may have explained why a bruit was readily detected by electronic auscultation in these subjects.

Our study had limitations. We recruited only 100 patients, but our sample size was restricted by the prevalence of bruits in the large FDS2 cohort. Nevertheless, FDS2 patients appear representative of people with diabetes in a large urban Australian community,¹⁵ and the similarities in demographic variables between those with carotid bruits who participated in the present substudy and the 33 who did not suggest that there was no significant recruitment bias. We did not perform simultaneous acoustic auscultation, as our aim was to assess use of the electronic stethoscope against reference ultrasonography. It is likely, however, that electronic capture of carotid sounds is more reliable based on the greater prevalence of bruits in FDS2 versus FDS1 of 6.1% versus 4.5%³ and in other studies directly comparing the two methods.^{11,12}

The present study suggests that electronic auscultation is more accurate than a conventional acoustic stethoscope for the detection of carotid bruits in patients with diabetes, while offering the convenience of later interpretation if a skilled operator is not available when the patient is assessed. The presence of a bruit identified in this way should prompt (1) intensified cardiovascular risk factor management and (2) consideration of ultrasonography and perhaps other imaging with a view to revascularization, depending on symptoms and factors such as age and comorbidities. The absence of a bruit does not exclude a hemodynamically significant stenosis, but there may be other indications of cerebrovascular disease. For example, in our patient without a bruit who had an occluded carotid artery, there was a history of cerebrovascular symptoms and evidence of established vascular disease at other sites that is in itself collateral evidence of significant carotid stenosis risk.^{10,34} The cost of the electronic stethoscope and software used in the present study is approximately twice that of a high-quality acoustic model, suggesting that it is affordable technology even in basic healthcare settings.

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Author Disclosure Statement

No competing financial interests exist.

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